A Strategy for Studying Differential Vulnerability to the Psychological Consequences of Stress

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It is important to examine the possibility that relationships between commonly studied status indicators and psychological distress indicators are due, in part or wholly, to differences in the impacts of comparable stressor events and situations on people in different status categories. This paper describes and illustrates a strategy for doing this and argues that the systematic existence of impact differentials helps explain the commonly observed relationships between certain social statuses and psychological distress.

There are a number of social stress models currently being used by researchers interested in mental illness (Scott and Howard, 1970). All of them, though, share the assumptions that (1) exposure to stress impacts on psychological distress, and that (2) various "resources" available to certain individuals modify the severity of these impacts. Most empirical sociological work based on these models has concerned itself with the first of these two assumptions (Dohrenwend, 1973; Dohrenwend and Dohrenwend, 1969; Myers et al., 1974). In this paper, by contrast, I describe a strategy for examining the second assumption.

Background

Over the last several decades many social epidemiological studies of psychological distress have found several consistent relationships between various distress measures and a number of social statuses (Dohrenwend, 1975; Dohrenwend and Dohrenwend, 1974a, 1974b). Although social class has been the most completely studied of these (Dohrenwend, 1973; Dohrenwend and Dohrenwend, 1969; Hollingshead and Redlich, 1958; Myers et al., 1974; Srole et al., 1962), others of interest include sex (Dohrenwend and Dohrenwend, 1976, 1977; Gove, 1972; Gove and Tudor, 1973, 1977), marital status (Bachrach, 1975; Pearlin and Johnson, 1977), and rural versus urban location (Dohrenwend and Dohrenwend, 1974a; Kasl and Harburg, 1975).

Recent work has concerned itself with interpreting the causal processes that lead to the observed relationships between social status and psychological distress. Major emphasis has been on the differential exposure hypothesis, which argues that an unequal distribution of potentially stressful life events and situations accounts for the unequal distribution of psychological distress among people in socially advantaged and disadvantaged statuses (Dohrenwend, 1973). The Dohrenwends (1969:53), for example, speak of social environmental influences among members of the lower class as "a function of the strength of the social pressures experienced by members of this class." Similarly, Gove's (1972; Gove and Tudor, 1973, 1977) sex-role theory of mental illness speaks of the great emotional "strains" experienced by women in our society and the great ambiguity of female roles.

Although a few empirical studies have reported results suggesting that important relationships between status and psychological distress can be explained by stress distribution (Dohrenwend, 1973; Myers et al., 1974), most work has failed to support this hypothesis (Markush and Favero, 1974; Pearlin, 1975; Pearlin and Johnson, 1977). Indeed, it has been

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shown in a number of studies that some social statuses are not even related to some life-events scales. Myers et al. (1974) found this to be true for all but one scale in their New Haven study of social class and mental illness. Markush and Favero (1974) and Holmes and Masuda (1974) both found sex to be unrelated to the life change units. Most studies show that the widowed, usually the most highly distressed marital status group, have fewer recent life crises than members of any other marital status group (e.g., Holmes and Masuda, 1974; Kessler, 1979).

It would seem, then, that differential exposure to life stresses cannot, in itself, account for the commonly observed relationships between social status and psychological distress. We simply find too many psychologically healthy people who have suffered extreme life crises, and too many psychologically distressed people who have experienced only their normal share of life problems, to claim otherwise. Not surprisingly, then, most recent models of the stress-distress nexus (e.g., Cobb, 1976; Rahe and Arthur, 1978), as a way of augmenting the stress distribution hypothesis, include the idea that some people are more "vulnerable" than others to the psychological consequences of stressful life situations. Indeed, in a recent review of this literature, the Dohrenwends (1974c:316) comment that

> for some investigators the central problem concerning the effects of stressful life events, grows out of the observation that one individual may become ill and another remain healthy after both experience the same life event. The most general formulation of the research question generated by these individual differences is: What are the factors that mediate the impact of stressful life events on the individual?

Unfortunately, more times than not the concept of differential vulnerability is used in a *post hoc* fashion to account for the failure of stressor measures to have the predictive power one would expect in regard to the psychological distress exhibited by people in normal populations. Only a very few empirical studies have used the concept as a way of better understanding the relationships between sociocultural factors and distress. Perhaps the most impressive of these to date, though, is the work of George Brown and his associates (1975), which found working-class women in London more likely to exhibit symptoms of clinical depression than middle-class women, *when the two were exposed to comparable stressor situations*. This finding demonstrated empirically an important class-linked impact differential. Indeed, Brown found that observed class differences in depressed mood could be completely accounted for by this differential impact, even though class differences in exposure to stressful situations were not strong.

Clearly, it is important to examine the possibility that relationships between other sociocultural factors and other indicators of psychological distress are due, in part or wholly, to group differences in the impacts of comparable stresses, and to explain how it is that these impact differentials come to be. To date, though, we have lacked an adequate conceptualization that could be readily used in conventional epidemiological research. In the next section of this paper I describe such a conceptualization and present an analysis strategy that allows one to describe such effects elegantly and simply. In the final section of the paper I discuss ways in which this approach can be augmented to allow one to study the determinants of vulnerability.

THE MODEL

As I conceptualize it, psychological distress (P) is the result of varying exposure to environmental stress events or situations (S) acting on individuals who possess varying vulnerabilities to stress (V). By *vulnerability* I mean the force with which a stress impacts on the distress of an individual. I do not mean to imply that only one or even a small number of determinants go to make up the force of impact, or vulnerability, that characterizes an individual's response to a particular stress. Indeed, as I discuss below, I believe that these vulnerabilities are both constitutionally and environmentally determined in a rather complex fashion.[1]

In a simple functional formulation we can express this relation as follows:

$$P_i = V_i (S_i) + a_i , \qquad (1)$$

where a_i represents the residual influence of

constitutional makeup on the mental health of person i independent of any environmental stresses he/she might experience. Alternatively, the two-part makeup of the impact coefficient might be made clear by explicitly recognizing the separate constitutional (V_c) and environmental (V_e) components, so that we have:

$$P_i = (V_{ic} + V_{ie})(S_i) + a_i . \quad (2)$$

If we think of objective stressors as potentially quantifiable and if we think of distress as countable in terms of some average symptom rating score, we can estimate V empirically by (1), assuming that P and S are measured for each individual in each contrast group. Then V becomes a regression coefficient.[2] On the other hand, if we use (2), this cannot be done, since there is no way to estimate V_c and V_e separately. There is a certain respect in which (1) is unappealing, for it introduces an ambiguity into the interpretation of V. However, it can be informative to begin an analysis of between-group differences in psychological distress with model (1).

As an illustration, Table 1 gives the results of two regression analyses estimated separately for men and women from the 1967–1969 New Haven community panel survey of 720 adults (Myers et al., 1972, 1974, 1975). The predictors here, all measured in 1969, are three life-events scales and two measures of ongoing stressor situations. These are: (1) an overall count of desirable life events experienced in a two-year period; (2) an overall count of undesirable life events experienced in the same time period; (3) an overall count of ambiguous events experienced in this time period (for more detail, see Myers et al., 1974:195–97); (4) a measure of economic stress, operationalized as total family income per family member; and (5) an overall count of physical illness symptoms, purged of those that are clearly psychosomatic.

The criterion measure of distress is the 20-item Macmillan (1957) symptom checklist scale as modified by Gurin et al. (1960). The scale is made up of questions about the respondents' perceptions of bodily feelings that indicate depression and anxiety as they relate to psychophysiological symptoms of distress. Scores on the scale range from 20 (all of the symptoms are reported as occurring for the respondent "often") to 80 (symptoms are completely absent).

Women score approximately 1.619 points higher than men on overall symptom scores, a small but significant difference. However, we can make the comparison more formally, using all the information in Table 1, by means of a demographic rate standardization technique (Winsborough and Dickinson, 1971), which, in the notation of (1), gives us:

$$\begin{aligned}(\bar{P}_m - \bar{P}_f) = &\sum_j (V_{mj} - V_{fj})\,\bar{S}_{fj} \\ &+ \sum_j (\bar{S}_{mj} - \bar{S}_{fj})\,V_{fj} \\ &+ \sum_j (V_{mj} - V_{fj})(\bar{S}_{mj} - \bar{S}_{fj}) \\ &+ (a_m - a_f) , \end{aligned} \quad (3)$$

where j = 1 . . . 5 (in this case, the five predictor variables), and where m = male and f = female.

The four components can be interpreted by using the imagery of an experiment. The first component represents the expected change in female symptoms if females were given the aggregate vulnerabilities of males. The second

TABLE 1. Regressions of Gurin Symptom Scale Scores on Measures of Life Events and Ongoing Life Situations, by Sex

	Males (N = 318)			Females (N = 402)		
Predictors	Slope	Mean	S.E.	Slope	Mean	S.E.
Undesirable events	−1.302	.739	.470	−2.358	.794	.331
Desirable events	.103	.396	.577	− .256	.522	.454
Ambiguous events	− .852	.837	.380	−1.028	.988	.307
Income/family member	.691	2.012	.256	.237	1.671	.262
Physical illness	−2.238	.767	.351	−1.690	.719	.325
Constant	75.698	—	—	75.956	—	—
R^2	.214			.222		

Source: Wave II (1969) New Haven panel data (Myers et al., 1975). Missing values are assigned means.

component represents the expected change in female symptoms if they were exposed to the stressful events experienced by males. The third component represents the expected change in female symptoms if they were simultaneously exposed to the stressful events experienced by males and also given the overall vulnerabilities of males. Finally, the last component represents the residual vulnerability differences unassociated with the stresses included in the model.[3] For simplicity we shall refer to these four components below as, respectively, the *impact, exposure, interaction,* and *residual* components. Together, these four components capture all the influences on male/female symptom differences modeled in the two regression equations.

Applying (3) to the equations in Table 1, we have the following component breakdown, expressed in units of the symptom scale:

Impact	=	1.564
Exposure	=	.317
Interaction	=	−.001
Residual	=	−.261

To put these values into words, women are disadvantaged relative to men both in being more vulnerable to the stresses they experience and also in being more exposed to stressful events and situations. Net of these two disadvantages, women are less symptomatic than men by a small margin.[4] However, the exposure, interaction, and residual components are very small.[5] By comparison, the impact component of 1.564 almost totally reproduces the observed 1.619 sex difference. For all practical purposes, then, the greater distress exhibited by women is due to the fact that they are more highly affected psychologically by stressful events and situations to which they and men are equally exposed.

In a full analysis of these data we would certainly not stop here, but would try to gain some understanding of the greater vulnerability of women. It is important to realize, though, that we need an unambiguous way of describing vulnerability before we can try to understand it. This is exactly what we achieved by conceptualizing vulnerability as an impact coefficient and by using a formal decomposition strategy to study impact differentials between contrast groups. With this much done, it becomes possible empirically to study the determinants of differential impact. In fact, in a more extensive analysis of these data,[6] two factors were found implicated in the greater vulnerability of women: (1) Financial resources in the family are less helpful to women than to men in alleviating psychological distress; and (2) women make use of coping strategies that are less effective than those used by men in buffering them from the psychological consequences of undesirable life events.

In the next section I discuss general strategies for interpreting impact components of this sort after they are uncovered in a decomposition.

APPROACHES TO THE INTERPRETATION OF IMPACT COMPONENTS

Broadly conceived, vulnerability differentials can be caused by three classes of factors: (1) There may be biological influences that make members of one contrast group constitutionally more fragile than members of the other group. (2) The environment may influence vulnerability in a number of ways. For one, it may influence the subjective meaning of objective situations, so that the same events are experienced as more stressful by members of one contrast group than the other. The environment may also provide social support or other types of resources to buffer the psychological consequences of stressful events. Finally, socialization experiences can be influential in supplying the individual with self-esteem or other personal resources for handling life problems. (3) At least two sets of methodological effects could be at work, leading us falsely to assume that a vulnerability effect is present. The first would appear if members of one contrast group systematically over- or underreported either symptoms or events, relative to the other group. Bias of this sort would have the effect of inflating or deflating the metric of the slope values for one or the other group, so that slope comparisons could not legitimately be made. The other methodological problem would appear if the researcher erred in his/her conceptualization of stressor dimensions, so that two events would be interpreted as the same even though they were very different in some important way. For instance, women might suffer more high-impact undesirable

events than men, but men suffer more low-impact undesirable events than women. Then a simple count of undesirable events experienced, without weighting the events in terms of impact, would show women to be more "vulnerable" to the "same" events than men. In actuality, though, this "vulnerability" effect would be masking an important sex difference in exposure.

There is no information in the decomposition analysis itself to argue for or against any of these possible influences. As a result, the researcher must bring outside data to bear on his/her contrasts to aid in understanding the impact component. Fortunately, the decomposition strategy lends itself to the use of outside data, in the form of control variables in the regression analyses. This is so because the strategy allows the analyst to divide the impact of each predictor into four additive components. If we introduce some predictors that are controls, then we can examine their influence in modifying the impact effects of the other measures, the stressors. In this way we can develop an understanding of the outside influences that modify the relative vulnerabilities of our contrast groups.

There are a great many specific types of control analyses that might be undertaken. I shall mention only a few of these here. (1) A body of work concerned with the meanings of potentially stressful events and situations is beginning to appear in the literature (Brown, 1974; Brown et al., 1973). Findings here have a great potential for helping us evaluate the hypothesis that vulnerability differences are due to between-group variations in the ways events or situations are experienced, by providing measurable distinctions among the important dimensions of meaning that modify the psychological impact of stressor events and situations. (2) Recent work by Pearlin and Schooler (1978) has gone far toward operationalizing measures of various coping strategies and resources. These measures, or others like them, might be used as controls in the decomposition analysis to help partial out vulnerability effects due to group differences in resources and strategies.[7] (3) A series of recent studies have examined the hypothesis that the female distress preponderance in psychiatric screening scales is due to the greater willingness of women than men to express their problems (Clancy and Gove, 1974; Gove and Geerken, 1977; Phillips and Clancy, 1970; Phillips and Segal, 1969). The naysaying-yeasaying and social desirability scales used as controls for this sort of bias could be used as methodological controls in the decomposition analysis.

In none of the control analyses suggested here do we consider the impact of biological factors that might make one set of individuals more vulnerable than another. Indeed, we lack so much as a clear idea of what biological factors to measure. However, there is one respect in which the decomposition approach is compelling even here. In those special cases in which the impact component is small and the exposure component is large, a case can be made for the fact that constitutional frailties have no impact on between-group differences in symptomatology. This is especially true when the measures of events and ongoing situations used in the equations cannot be attributed to prior symptom-proneness. This interpretation is far more plausible in such special cases than the chief contending interpretation, that constitutional advantages and environmental disadvantages cancel each other out.[8] This latter interpretation, in fact, would be even less plausible in contrasts between, say, middle- and lower-class whites, for here we could plausibly argue that the constitutional and environmental forces that modify the impact of stress on distress probably both work to the advantage of the middle class. Thus the possibility that these two separate sets of impact modifiers cancel each other out (which implies that they work in opposite directions) is called into question.

Although it is unlikely that a trivially small impact component of this sort will appear in most theoretically interesting comparisons, a combination of the use of control variables and use of the above conceptual argument is possible. That is, if we can account for the impact component by introducing a series of controls for environmental influence that cannot plausibly be attributed to a possible biological influence, we can reasonably argue that a biological component in group differences is absent.

Discussion

An appealing feature of the strategy just presented is that it does not require any special

changes in the sorts of data normally collected in community surveys. Indeed, the strategy requires only a small shift in the way stress-distress data are normally analyzed. There is no reason, then, for researchers not to examine their data for the possible existence of impact differentials when studying relationships between sociocultural factors and psychological distress.

Thus far I have focused on a regression-based approach to the comparison strategy, which treats the distress measure as an interval scale. This might have given the false impression that the general strategy is tied to linear model theory. Rather, the key features of the approach I advocate are (1) the use of an impact coefficient to represent vulnerability and (2) the comparison of these coefficients between contrast groups to represent differential vulnerability. These features can be incorporated into a model for categorical variables. Following the lead of Srole et al. (1962) and Myers et al. (1974), we can define symptom scale scores above some cut-off point as "cases." When this is done, we can use a logit model to predict the odds of a respondent being a case or not (Nerlove and Press, 1973). The decomposition between two groups is then a multiplicative one, based on the odds-ratio (OR) of distress/non-distress between the contrast groups. That is, if one thinks of the vulnerability, exposure, interaction, and residual components in (3) as t, u, v, and w, respectively, the logit decomposition becomes:

$$\mathrm{OR} = e^{t} \times e^{u} \times e^{v} \times e^{w} . \qquad (4)$$

Using this general formula, one can define an impact component unambiguously even when the distress measure is categorical, and the stressors and controls are either categorical or interval.[9]

To date, the dominant emphasis in stress-distress research has been on the differential distribution of stressors across sociocultural groups. The availability of a general conceptualization of vulnerability makes it possible now routinely to broaden this focus and to study the relative importance of the differential distribution of stress and the differential impact of stress in accounting for sociocultural group variations in psychological distress. I believe that this should be done, for the differential exposure hypothesis is not, by itself, powerful enough to account for the preponderance of psychological distress among women, the poor, and the unmarried. Indeed, in analyses of the New Haven data reported elsewhere (Kessler, 1979) it is shown that consideration of impact differentials is necessary for a clear understanding of the relationships between distress and the factors of sex, income, and marital status in those data. It is probably true, to some extent, that people in disadvantaged positions in society are exposed disproportionately to stresses; but at least as important is the fact that these people are more highly affected than others by the stresses to which most people are exposed. It is not clear why this is the case, but I believe that research efforts should be directed toward uncovering the sectors of people in society who are more vulnerable than others and toward understanding the causes of this differential vulnerability. The strategy presented here can be extremely useful on both these fronts.

NOTES

1. I will not discuss the complex methodological problems associated with (1) the use of treatment statistics as definitions of disorder; (2) defining a "case" or degree of "symptomness" in epidemiological surveys; (3) defining the experienced "stressfulness" of objective stressor events. These are all very real and important problems that must be considered by the researcher. However, I have nothing to add to the lucid discussions that exist about these problems in the literature.
2. We assume that each individual has his/her own degree of vulnerability and we would ideally prefer to assign vulnerability scores on an individual basis. However, since we must infer the vulnerability coefficient as a parameter in our model, we can estimate it only as a constant across a sample of individuals. In order to evaluate the relative importance of impact and exposure then, we are forced to compare two samples, thus obtaining a variation in vulnerability. This leaves us in the position of having only an aggregate-level analysis rather than one on the individual level; but this is necessary as long as we lack adequate measures of constitutional fitness for individuals.
3. We can interpret this component as the effect of constant stresses associated fundamentally with the sociocultural group we are examining through comparison. For instance, we might argue along with Gove that women as a group experience certain common stresses that are not within the experience of men, thus causing women, as a group, to exhibit a constant increment in symptomatology over men.

In general, the decomposition procedure assumes that there is enough variation in the experience of stress within each contrast group to allow estimation of a stable vulnerability coefficient. To the extent that we believe this is not true, the residual must be considered as a possible constant stress effect. We would like to introduce sufficient measures of stress and controls into our models to account totally for residual effects, allowing us perfectly to account for mean symptom differences across the contrast groups. This would, of course, deal effectively with the problem of interpreting the residual component. In practical terms, though, some relatively constant and unmeasured role strains probably do exist in different subpopulations. It is, therefore, unrealistic to hope that intercept differences will be explained by controlling explicit stressor variables. Nor is it likely that we will be able to measure and control explicitly all the various stressors that differ across two subgroups. For both these reasons we can expect that intercept effects and the ambiguities they represent will appear in most empirical research using this approach.

4. Robert Hauser pointed out to me that global tests should be made to test the possibilities that either one or both sets of differences are insignificant in statistical terms. Clearly, one would not want to interpret components based on randomly varying coefficients. A general approach for assessing the significance of the decomposition has recently been developed by Michael Sobel, at the University of Wisconsin. However, this approach has not yet been programmed and requires extensive hand calculation. An alternative procedure is to proceed in two steps. First, evaluate the significance of mean differences for each predictor across the two subsamples by means of t-tests. Second, evaluate the significance of slope differences by means of pooled regression models containing a dummy variable for sample membership (in our case, D = 1 for men, 0 for women) and interaction terms for the stresses (S) of the form D×S.

   Applying this approach to the sex example, we found that no mean differences are significantly different from zero and that only the slope difference associated with undesirable events is significantly greater for women than men. If we use this information as the basis of a decomposition, an impact component of .827 and a residual component of .792 are estimated. The exposure and interaction components are assumed to be zero, since each is based on a product of mean differences. The equation on which this decomposition is based is (significant coefficients starred):

$$\begin{aligned}\bar{P} = {} & 75.588 - 2.334^{*}\,(\text{Undesirable events}) \\ & - .129\,(\text{Desirable events}) \\ & - .961\,(\text{Ambiguous events}) \\ & - 1.194^{*}\,(\text{Physical illness}) \\ & + .475^{*}\,(\text{Income/Family member}) \\ & + .492\,(\text{Dummy male}) + 1.042^{*}\,(\text{Dummy} \times \text{Undesirable events})\end{aligned}$$

5. The interaction term is trivially small and can be assumed to be of no importance. The interaction effect, in general, however, can be interpreted as we would an overlap exogenous effect between two predictors in a linear, recursive model formulation; that is, a measure of the empirical confounding of two effects. For various technical procedures for dealing with interactions in the algebraic formulation, see Iams and Thornton (1975).
6. The results of this more complete analysis are available from the author on request.
7. This type of control analysis requires the use of interaction terms in the regression models, to represent the hypothesis that stressor impacts vary as a function of the utilization of varying types of coping strategies and resources. The Pearlin and Schooler (1978) analysis failed to include interactions of this sort, thus making it difficult to accept their interpretations about the relative effectiveness of the various strategies and resources they examined. I have profited from a number of useful conversations with Paul Cleary concerning this point.
8. This interpretation can be made, though, only when the interaction component is also small. If interaction is large, on the other hand, a small impact component can be representing the collinearity of impact and exposure in the contrast, rather than the absence of a significant impact component.
9. The logit model has the following form:

$$\ln\left(\frac{p_i}{1-p_i}\right) = V_i\,(S_i) + a_i\,,$$

   where p represents the predicted probability that person i will be a "case." This equation is linear in log-odds, so that we can use (3) to decompose the difference in log-odds between two contrast groups. That is,

$$\ln\left(\frac{p_a}{1-p_a}\right) - \ln\left(\frac{p_b}{1-p_b}\right) = t + u + v + w$$

   for contrast groups a and b. By taking the antilogarithm of this equation we arrive at (4).

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Social Support, Stressful Life Events, and Illness: A Model and an Empirical Test

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The effects of social support and stressors (stressful life events) on illness (psychiatric symptoms) are examined in a model with data from a representative sample of the Chinese-American adult population in Washington, D.C. The analysis shows that, as expected, stressors are positively related to the incidence of psychiatric symptoms, and social support is negatively related to psychiatric symptoms. Further, the contribution of social support to predicting symptoms is greater in magnitude than that of stressful life events. When marital status and occupational prestige are incorporated into the model, the significant (negative) contribution of social support to symptoms is not reduced. Implications for the theoretical development of a sociomedical theory of illness are discussed.

This paper examines the potential role of social support in relation to stressful life events and subsequent illness. Two of these parameters of epidemiology, stressful life events and illness, have been amply explored in recent epidemiological literature. The third, social support, while being considered in numerous discussions to be related to the incidence of illness, has not been systematically linked to the stressor-illness model.

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After a brief review of the literature relating stressful life events to illness, we shall discuss the potential theoretical significance of social support in the study of the etiology of illness. A proposed model is then tested using data gathered from a Chinese-American population.

In the last 15 years, one important thrust in the study of the etiology of illness has been the examination of the potential causal effect of life changes on illness. The pioneering work in this area by Meyer (Lief, 1948), Holmes, Rahe, and their associates (Holmes and Masuda, 1974; Holmes and Rahe, 1967; Rahe, 1975; Rahe et al., 1964) led to the development of life-events scales. High scores on life-events scales (from either the number of events or the sum of